LARYNGEAL AND PHARYNGEAL PARALYSES.

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I ASK attention to a consideration of paralysis as affecting the muscular structures of the pharynx and larynx. In order that I may bring forward the question whether we are not, in the large proportion, if not in all cases, to consider true neuropathic paralysis in these regions an evidence of a lesion of the medulla. Of course we exclude cases of myopathic paralyses such as follow diphtheria, as well as those cases of recurrent laryngeal paralysis dependent on pressure on the nerve trunk.

That classification of laryngeal paralyses which is based entirely on the anatomy of the organ, such as is given in Mackenzie's[1] earlier work and followed later by myself,[2] must be abandoned as failing to make any distinction between true paralysis and myopathic conditions, such as simple muscular weakness.

In this manner our interest in laryngeal paralysis becomes centered really in two conditions, viz., unilateral or bilateral paralysis of the abductor and recurrent laryngeal paralysis. The consideration of pharyngeal paralysis is added as contributing weight to the view that the true clinical significance of either form is in the suggestion that it is due in most instances to some lesion of the medulla.

While the whole motor innervation of the larynx is derived from the recurrent laryngeal nerve, with the exception of the crico-thyroid muscle, which in the present consideration may be discarded, we find this organ endowed with two apparently separate and distinct functions, viz., that of phonation and respiration, the movements by which these functions are accomplished being directed apparently through the same nerve trunk, yet possessing an action each so entirely independent of the other that they afford a subject of study from a physiological point of view of no little interest, and notwithstanding the many and able contributions which have been made to literature on this subject, it seems to me there are many questions which still remain unsolved, especially those connected with paretic conditions of the respiratory function, namely, unilateral or bilateral paralysis of the abductor muscles. That this disease occurred before the days of laryngoscopy cannot be questioned, yet a diagnosis can only be established by a physical examination; hence, our recognition of the affection dates from comparatively recent times, and the disease still is an exceedingly rare one. Mackenzie, in his first edition reporting but a single case, while Ziemmsen[3] had collated nine cases. Mackenzie[4] in his second edition reports having observed sixteen cases, while in a study of the subject made by myself in 1881,[5] thirty cases were collated. In the same year Semon[6] made a valuable contribution to the subject based on an analysis of twenty-two cases. In addition to this, valuable contributions have been made by Krause,[7] Eisenlohr,[8] Sokaloff,[9] B. Frankel,[10] Jean,[11] Senator,[12] Biermer[13] (as cited by Gottstein), Oppenheim,[14] Ollivier d'Angers[15] (as cited by Gottstein), Gerhardt[16] (as cited by Gottstein), Stephen Mackenzie[17] (cited by Gottstein), and Saundby.[18]

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[1] Diseases of the Throat and Nose, N. Y., 1881.

[2] Laryngoscope, Hoarseness and Loss of Voice. Am. ed., Philadelphia, 1869.

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[3] Cyclopedia of Prac. of Med., N. Y., 1876. vol. vii., p. 959.

[4] Diseases of the Throat and Nose, Amer. ed., 1880, vol. i., p. 442.

[5] N. Y. Med. Jour., Nov., 1880.

[6] Arch. of Laryngology, vol. ii., p. 197.

[7] Neurologische Centralblatt, 1885, p. 543. Berlin Klin. Woch., 1886, No. 20, p. 651. Journal of Laryngology, July, 1888.

[8] Deut. Med. Woch , 1886, No. 21, p. 363. Arch. f. Psychiatrie, vol. ix., p. 1–45.

[9] Deut. Arch. f. Klin. Med., vol. xli., p. 458.

[10] Berlin. Klin. Woch., 1886, No. 40, p. 675.

[11] Progres Medicale, 1876, No. 20.

[12] Arch. f. Psychiatrie, vol. xi.

[13] Die Krankheiten des Kehlkopfes. Leipsig und Wien, 1888, p. 305.

[14] Berlin. Klin. Woch., 1886, No. 40, p. 675.

[15] Op. cit., p. 309.

[16] Op. cit., p. 310.

There are three views which have been advanced to account for this curious disease:

I. That it is due to a peripheral lesion;
II. That it is due to a morbid lesion in the continuity of nerve; and
III. That it is due to a central lesion.

I. As regards the peripheral lesion: Mackenzie[19] in his earlier work states that the cause of the disease is generally cerebral, "but morbid influences which affect both pneumogastric or both recurrent nerves may give rise to it," while in his later work[20] he makes the suggestion that owing to the exposed situation of the abductor muscles on the posterior aspect of the larynx they are especially liable to become the seat of injury, which added to the fact of their ceaseless activity renders them vulnerable, and suggests that this fact may act in a causative relation to the disease, apparently abandoning the idea that the disease may be of cerebral origin. Following the same line of argument, Smith[21] explains the increasing dyspnœa, which apparently occurred in his reported case by the suggestion that the progressive weakening of the posticus muscles resulted in a prolapse, as it were, of the posterior wall of the larynx. It may be interesting to note, in this connection, that Smith's case subsequently suffered an attack of cerebral hæmorrhage. Gowers,[22] in an ingenious course of reasoning points out the fact that the abductor muscles are inserted into the artenoid cartilages at an acute angle, while in the adductors this insertion is at a right angle. Hence, pressure affecting the nerve-trunk results in a paralysis, first of the abductors and subsequently of adductor muscles. Krause, in his admirable series of articles on this subject, seems to have reached the conclusion that the disease is due to a spasm of the adductor muscles, this spasm being due either to a central lesion or is the result of reflex irritation caused by a lesion of the superior laryngeal nerve. I think it is noticeable that when we attempt to explain the phenomena of bilateral paralysis by a diseased condition of the muscular structures themselves we are dealing largely with pure theory or speculation rather than the direct teaching of clinical observation. A close study of the cases on which this theory is based it seems to me hardly justifies the view that a simple local lesion explains all the phenomena of the disease. Thus, in Smith's case the cerebral hæmorrhage which subsequently occurred, points very emphatically to a pre-existing cerebral disease, which giving rise in the first instance to paralysis of the abductors subsequently developed an apoplectic seizure. Furthermore, I recall no well-authenticated case of recovery from bilateral paralysis of the abductors, whereas simple myopathic paresis is not usually regarded as necessarily incurable. Still further it should be stated that the onset of abductor paralysis is invariably sudden and without warning, whereas a muscular paresis in the nature of the case must be slowly progressive. Krause's curious theory of muscular spasm is open to the same objection, I take it, as that of myopathic paresis, and furthermore still leaves us somewhat in the dark. No one has, as yet, demonstrated what constitutes muscular spasm. Certainly, as regards laryngeal spasm, I am strongly of the opinion that this chapter of laryngology is still to be written by a wiser hand than has yet undertaken the subject.

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[17] Op. cit., p. 319.

[18] Birmingham Med. Review, Dec , 1886.

[19] Op. cit., p. 214.

[20] Op. cit., p. 437.

[21] British Med. Journal, July 13th, 1878.

[22] Lancet, June 5th, 1886, p. 1077.

II. As to a morbid condition of the continuity of the nerve as a cause of the disease. Pressure on the recurrent laryngeal nerve or other morbid conditions which may interrupt the nerve current has been a favorite theory by which the phenomena of abductor paralysis is explained. The advocates of this theory are immediately met with what seems to me an insurmountable objection. In a former article on this subject I made this statement. "As to the suggestion that the disease is due to pressure on the recurrent nerve, and that this pressure so far discriminates between the nerve fibres as to destroy the conductivity of those fibres alone which are distributed to the abductor muscles; it seems to me that the assertion is utterly untenable; that this might happen on one side alone, and that a tumor pressing upon the trunk of the recurrent nerve might so far select its point of pressure as to paralyze the abductor muscles on that side is among the possibilities; that this should happen on both sides and to both recurrent nerve trunks would be one of the rarest of coincidences; that it should happen in a large series of cases is simply beyond the pale of possibility." In one of Semon's cases pressure on the recurrent laryngeal nerves seems to have resulted subsequently in bilateral paralysis of the abductors. Here is a clinical observation which, it would seem, is exceedingly difficult to harmonize with the above assertion. The investigations of Exner and Weinzweig,[23] however, enable us to explain this case of Semon's, which, by the way, I think is quite unique. Both these observers have shown a crossing of the nerves of the larynx, especially of the superior laryngeal, by which the muscles of one side receive innervation from the nerve trunk entering the opposite side, while Mandlestam[24] has demonstrated that some of the fibres of the adductor muscles receive innervation from the superior laryngeal nerve. Ziemmsen[25] makes the statement that "paralysis of individual branches of the recurrent nerves may arise from incomplete lesions of the trunk as the result of unequal pressure, or when the nerve fibres are affected in an unequal degree by degenerative changes." Semon[26] completely endorsing Ziemmsen's statement, goes further and asserts "there is a distinct proclivity of the abductor fibres to become affected in such cases either at an earlier period than the adductor fibres or even exclusively." This proclivity-theory, so ably advocated by Semon, had already been advanced by

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[23] Monatschrift f. Ohrenheilkunde, Dec., 1884.

[24] Monatschrift f. Ohrenheilkunde, Dec., 1884.

[25] Op. cit., vol. vii., p. 948.

[26] Loc cit.

Rosenbach[27] and based on the observation of a single case, and subsequently sustained at the London Congress.[28] I find it difficult to see wherein this idea of Semon's adds to our previous knowledge of the disease. What does he mean by proclivity? What is the essential anatomical or histological condition wherein lies this proclivity or constitutes a proclivity? It seems not unfair to suggest an analogy between this and that definition of the action of opium as producing sleep by its hypnotic powers. Moreover, if Semon endorses Ziemmsen's teaching that the individual laryngeal muscles may be paralyzed by unequal pressure on the nerve trunk or differentiated degenerative changes, we need seek no further for an explanation of the fact of the paralysis. The proclivity-theory becomes entirely superfluous and uncalled-for, if it is possible that the individual fibre of the nerve trunk which supplies the posticus muscles has been sought out and destroyed by any morbid process. I have no desire to ignore the teaching of the admirable series of experiments performed by Semon in connection with Victor Horseley,[29] although I do not think they sustain Semon's proclivity-theory. Briefly, these experiments were as follows: The recurrent laryngeal nerve in one of the lower animals was exposed and ligatured. At the end of several days the animal was killed, and examination showed degeneration of the abductor muscles, with but slight changes in the other muscles supplied by the nerve. In addition to this, another experiment was performed, by which the larynx was removed from the animal and the trunk of the recurrent nerve subjected to an electric current, demonstrating that the contraction ceased in the abductor muscles before the others, or, in other words, the abductors died first. The experiments of F. Donaldson, Jr.,[30] in connection with Prof. Martin in the laboratory of the Johns Hopkins University, differed from the above in that they were performed upon a living animal, although subsequently verified on the dead subject. The conclusions were virtually the same, in showing that as the result of stimulation of the recurrent laryngeal nerve by the electric current the adductor muscles survived the abductors. The admirable series of experiments made by Hooper,[31] in connection with Prof. Bowditch, in the physiological laboratory of Harvard University, in the same line of investigation failed to substantiate the conclusions arrived at by Semon, Horseley and Donaldson, in that under electrical stimulus the abductor fibres showed an amount of vitality fully equal to the adductors. The fact, however, seems to be established beyond dispute that under stimulation the adductor fibres show greater vitality than the abductors, or, in other words, that the abductor fibres show a proclivity, if you like, to paralysis, Hooper's experiments alone failing to establish this view. This greater vitality of the adductor muscles is still further illustrated in those cases of bilateral anchylosis of the crico-aritenoid joint, improperly regarded as cases of bilateral paralysis of the abductor muscles. Thus in the two cases reported by Lefferts[32] as the result of syphilis there was apparently bilateral paralysis. It seems to me, however, that a nicer diagnosis would show that the blood-poison had given rise to a local anchylosis of the crico-arttenoid joint and fixation of the cords in the median line due to the fact that the more powerful structure of the adductor muscles overcame the less resistance on the part of the abductors.

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[27] Breslau Zeitschrift, 1880, Nos. 2 and 3

[28] International Med. Congress—Trans., vol. iii., p. 222.

[29] Brit. Med. Journal, 1886, Aug. 28th and Sept. 4th, pp. 405 and 445.

[30] Trans. of the Amer. Laryngological Assoc., 1886 and 1887.

How far these experiments throw light on the causation of the abductor paralysis I think still remains an open question. That they prove a difference in the response to electrical stimulation, on the part of the phonatory and respiratory muscles of the larynx, is conceded, and I think we may naturally expect this would be the case. The two functions differ essentially in a very marked degree. The respiratory movements of the larynx are unceasing, commencing with the first breath of life and only ending with dissolution. The phonatory movements of the larynx, on the other hand, are intermittent. The respiratory movements are involuntary, the phonatory movements voluntary. These functions, therefore, necessarily involve certain differences in innervation. I think an analogy may be suggested between the posticus muscles and the cardiac muscles, in that their contraction never ceases during life. May we not venture a further analogy in that, under an electric stimulus, the heart is arrested in dyastole, or, in other words, it is for the time paralyzed. Under the electric stimulus, if continued, the abductor muscle is paralyzed. The analogy, of course, is not complete, but a certain similarity in the action of these two muscles, the cardiac and the abductor, under the electric stimulus, is suggested.

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[31] Trans. Amer. Laryngological Assoc., 1885, '86, and '87.

[32] N. Y. Med. Journal, Dec., 1878.

After all, it is a question as to how far physiological experiment aids us in the elucidation of the finer points in neuropathology. Can the nice differentiation and delicate localization of a morbid process at the ganglionic centres, or in the continuity of a nerve, be in any way imitated, or even approached, by the harsh, coarse, and rude manipulations of the physiologist's scalpel and scissors? In clinical study, then, and analysis of symptoms as they present to us in the cases which come under our observation, I think we must seek for the true cause of these affections; and certainly, if my conclusions shall be correct, we will find a stronger confirmation here in the view that abductor paralysis is due not to a peripheral lesion or to disease of the nerve trunk, but to

III.—Some morbid condition of the nerve centres.

Mackenzie, in his later work, seems to have abandoned the idea that this disease was due to a central lesion, as suggested in his earlier work. In my article, already quoted, I wrote as follows: "Reasoning from analogy, considering the peculiar character of the respiratory movements of the larynx in that they are purely involuntary and also reflex, that the opening of the glottis constituting the respiratory movement is an independent action separate from all the other movements which take place in the larynx as the result of muscular contractions, it is fair to conclude that this function is presided over by an independent ganglionic nerve centre, and that the disease in question consists in some degenerative change taking place in this portion of the nerve centres."

This article was written in 1880, and I think that such light as has been thrown on the subject since that time only serves to substantiate this teaching. Furthermore, as before suggested, our best information must come from the study of cases and analysis of their symptoms. The following cases, hitherto unpublished, are taken from my own note-books.

CASE I.—Bilateral paralysis of the abductors in the early stage of tabes.

H. S. W., aged 48, merchant, was referred to me, June 5th, 1883, by Dr. Crumm, of South Otslich, N. Y., with the following history. In October, 1881, as the result of exposure, he contracted a cold, resulting in sore throat and loss of voice. At the end of a week he had a violent attack of dyspnœa, lasting perhaps a minute, characterized with noisy inspiration. The sore throat and loss of voice disappeared, but during the next fifteen months he had almost daily recurrent attacks of inspiratory dyspnœa, lasting a few second at a time, with no other symptoms. In addition to this he had occasional attacks of neuralgia, with some impairment of general health, otherwise no change. When he consulted me, a chest examination revealed nothing abnormal, in the larynx the mucous membrane fairly healthy, while the vocal cords were nearly motionless and parallel in the median line. I gave an unfavorable opinion on the case, and sent him home. After leaving New York he gradually developed motor and sensory paralysis, beginning in the right lower extremity, then affecting the left, and ultimately the upper extremities. These symptoms began in the winter of 1883–4, a few months before his death, which occurred in the spring of 1885, with marked evidences of paralysis of the faucial and lingual muscles.

CASE II.—Unilateral paralysis of the palatal and pharyngeal muscles as the result of bulbar hæmorrhage.

H. L., German, aged 63, merchant, consulted me on the 19th of August with the following history. Three months before, he arose in the morning with a feeling of general malaise, weariness on exertion, with a notable difficulty of articulation, and the feeling of an exceedingly thick tongue. On attempting to swallow at breakfast, the bolus lodged in his throat, giving rise to a threatened attack of suffocation until the mass was expelled. Fluids passed to the stomach slowly and with difficulty. The condition improved somewhat and until he consulted me, when he was able to take a fair amount of nourishment, although always with exceedingly great difficulty. An examination of the fauces showed the uvula slightly tilted to one side, while, on protrusion of the tongue, the tip was bent to the same side. His speech was slow and articulation thick. On pricking the pharynx and palate on one side, reflex contractions were excited with considerable difficulty, while on the other side the response was prompt. Nothing was noticeable in his movements in walking other than that they were slow and apparently feeble. On directing him to hop on one leg and then on the other, it was ascertained that it was impossible for him to stand or hop on the left leg. Treatment in this case was absolutely of no avail. The disease progressed slowly until the following spring, when he died of inanition. During the last three months of his life the only nourishment he received was administered through an œsophageal tube, deglutition being impossible. My diagnosis in this case was hæmorrhage into the medulla near the nucleus of the glosso-pharyngeal, pneumogastric, and hypoglossal nerves, with subsequent softening, invading the restiform and olivary bodies.

CASE III.—Paresis of the palatal and pharyngeal muscles as the result of obliterating endarteritis in a gouty patient.

H. M., aged 62, was a case similar to Case II., with the exception of the ultimate recovery. He consulted me, November 14th, 1886, with the history of rising in the morning a week previously with a feeling of general malaise and weekness of the limbs, which compelled him to lie down again. His speech was slow and thick, and articulation difficult. On his breakfast being brought to him in bed, he found considerable difficulty in swallowing both solids and liquids, although he managed to pass the softer kinds of food into the stomach, while the liquids were reguritated into the nose. An examination of this patient when I saw him, revealed the same symptoms as those of case No. II. The uvula was bent to the left, narrowing the palatal arch to that side, while the right arch was broader than normal. On protrusion of the tongue, the tip was turned to the right side. In attempting to stand or hop on one leg, a notable weakness of the left side was ascertained. This patient always enjoyed fairly good general health with the exception of occasional attacks of gout from which he had been a sufferer many years. This patient was under my care for two months, and made a fairly good recovery under the administration of general tonics, strichnia, and lithia, although even up to the present time there is a noticeable impairment in his articulation and lack of vigor in locomotion. The diagnosis in this case was obliterating endarteritis of one of the small vessels distributed in the medulla, undoubtedly the result of the gouty habit.

CASE IV.—Paralysis of the pharyngeal constrictors as the result of endarteritis deformans.

A. S. R., aged 52, merchant, consulted me, May 9th, 1888. Up to ten weeks before he consulted me he was perfectly well. On rising in the morning he found that he could not speak. There had been no promonitory symptoms, no giddiness, headaches, etc. On attempting to eat, he found that he could not swallow easily. Solid food seemed to stick in the throat and cause choking. There wâs slight weakness of the right hand, but no other paralytic symptoms of the extremities. He immediately began to be troubled with salivation, the tongue felt swollen, the lips could not be approximated to whistle or to say O. After this he went to Washington and Fortress Monroe, and seemed to improve under the warm weather. About a week before he consulted me he took cold, and the hoarseness, which had almost totally disappeared, and his difficulty in swallowing, which during this interval had not troubled him, returned. At the time of examination both symptoms seemed ameliorated, but still present. Since the attack he had lost flesh and strength. There were no cardiac, renal, or pulmonary symptoms. On examination, the larynx was normal, no difference in sensation between the two sides of the palate ;pupils sluggish to accommodation, react perfectly to light. No loss of power in the extremities, tendon reflex normal; no facial paralysis, no deviation of the tongue; no intrathoracic trouble found on physical examination. Retinæ normal, field of vision not limited. There was sluggish movement of the palatal and pharyngeal muscles on both sides. This patient was put under the administration of strichnine, and directed to return to Richmond and Washington for two months and abandon any idea of attending to business. At the end of this time I saw him again, and discovered a very satisfactory state of improvement, although there was still evidence of mild faucial paresis. This improvement has slowly continued, and he has assumed charge of his business in Maine. My diagnosis in this case was deforming endarteritis, probably the result of a gouty habit, there being no reason to suspect syphilis. The prognosis given was a guarded one, and made dependent on the careful regulation of his mode of life in the future.

The above cases are reported as containing many points of interest and offering much of suggestion in demonstrating the localized manifestations of the symptoms which arise from organic disease of the medulla. The next case is of special interest as illustrating how a localized morbid process in the medulla may give rise to both a laryngeal and pharyngeal paralysis.

CASE V.—Mrs. B., aged 53, consulted me, June 1st, 1888, with the following history. Several weeks ago she awakened somewhat suddenly in the morning with a violent cough, with difficulty in breathing, the difficulty being of an inspiratory character. In connection with this, there was moderate hoarseness and difficulty in swallowing. The dyspnœic attacks recurred daily, lasting a few seconds to a minute, and always characterized by a peculiar crowing inspiratory sound. These symptoms persisted without much change until I saw her, the dysphagia being of such a character as to seriously impair general nutrition. The hoarseness improved somewhat, but there was a noticeable impairment in phonation; the voice was weak. When I examined her I found the vocal cord of the left side motionless in the median line, indicating complete abductor paralysis; while on the right side there was a very limited amount of motion, abduction being but partially accomplished. An examination of the fauces showed sensation normal, but a very sluggish reflex action on both sides in responding to impact of the probe, more marked on the left side. Speech slow and somewhat difficult, not scanning. No deviation of the tongue. Stands easily with the eyes closed; touches the tip of the nose and ears with the eyes closed. No paresis of the muscles of the extremities. Tendon reflex normal. There was a suspicion of syphilis in this case which indicated the administration of iodide of potassium. The result of treatment was in every way satisfactory. At the end of a month the cough, dyspnœa, and dysphagia had completely disappeared, although an examination of the larynx showed the left vocal cord still motionless, with an improvement of the movements of the right cord, while the faucial muscles also showed an equal degree of improved action. With the exception of a slight relapse in the following week, the further history of this case was one of improvement, although on the last examination there was still complete left abductor paralysis.

CASE VI.—J. H., aged 33, saloon-keeper, was referred to me by a Dr. Highland, of Amsterdam, N. Y., on October 3d, 1887, with the following history. Fifteen months ago he awakened in the morning with almost complete loss of voice, with no notable symptoms other than some rather obscure neuralgic pains with difficulty or sluggishness of movement in his left arm. These symptoms persisted for a few weeks and gradually disappeared, involving no notable discomfort or interference with his ordinary occupation. One year later he was suddenly seized with an attack of what he describes as choking, a sudden catching for his breath, followed by loss of consciousness which lasted an hour. On emerging from this state, he was conscious of no new symptoms. His weak voice persisted and was perhaps a little more noticeable. At the suggestion of Dr. Hyland he consulted me on a casual visit to the city, at which time an examination revealed complete paralysis of the left side of the larynx, the cord remaining motionless in a cadaveric position. A thorough physical examination failed to reveal any discoverable lesion of the thoracic organs and no impairment in the organs of locomotion or speech. In this case there was probably an embolic formation in a small blood-vessel in his earlier attack, with a similar formation later, shutting off the blood-supply from a larger tract of the medulla. The interesting feature of this case lies in the fact that we have a bulbar lesion involving only the nuclei of those fibres of the accessory which go to make up the recurrent laryngeal nerve. That the lesion in this case was in the medulla I think can hardly be questioned in view of the fact of his second apoplectic seizure. This is notably substantiated also by many cases found in the literature of the subject, although I recall none reported in which the bulbar disease gave rise to recurrent laryngeal paralysis alone without involving other parts. Thus, in an interesting case reported by Hughling Jackson,[33] there was paralysis of the right side of the tongue with wasting paralysis of the right vocal cord, slight weakness of the limbs, especially on the right side. There was no post-mortem. The lesion was located by Dr. Jackson in the bulb. In another case by the same author,[34] there was sudden paralysis of the tongue on both sides, difficulty of deglutition, and loss of voice, with apparent paresis of the cords. There was no post-mortem. In this case, Dr. Jackson considered the diagnosis of bulbar disease as established. In another case,[35] there was paralysis of the seventh nerve, of the left half of the tongue and palate, and of the left vocal cord. Post-mortem examination showed softening of the medulla in the region of the anterior pyramid and nodes of the cerebral arteries. There was a history of syphilis in this case. In another case of the same author,[36] there was paralysis of the sixth, seventh, and eighth nerves of one side, the right cord being in the cadaveric position. Post-mortem examination revealed extensive disease in the right side of the medulla and pons. There was also a history of syphilis in this case. In the same connection, a case is reported by Proust, as cited by Hallopau,[37] of a female, aged 68, who suffered from complete paralysis of the vocal cords and dysphagia, intelligence not being affected. The autopsy showed thrombosis of the left vertebral artery, obliteration of the posterior inferior cerebellar artery, and softening of the floor of the fourth ventricle in the region of the nucleus of the hypoglossal, accessory, and facial nerves. In a case reported by Senator,[38] there was difficulty of swallowing, sensory paralysis, impaired voice with nasal intonation. Laryngoscopical examination showed double recurrent paralysis of the vocal cords. The autopsy showed an area of softening involving the olivary body, nucleus of the hypoglossus, and a portion of the fibres of the vagus. In a case reported by Smith,[39] there was inspiratory dyspnœa. Laryngoscopy showed double abductor paralysis, the patient a short time after having an apoplectic attack with loss of consciousness and slight impairment of power in the upper and lower extremities of the left side. Smith attributes the laryngeal symptom to exposure to cold and over-exertion; but from the subsequent history of apoplexy, it seems quite possible that the laryngeal symptoms may have been due to a bulbar hæmorrhage. Biermier, as cited by Gottstein,[40] reports a patient, aged 56, who suffered from recurrent paralysis of the right side, together with paresis of the right half of the palate and analgesia of the mucous membrane of the right side of the mouth and of the integument about the right eye. The attack came on suddenly and was attended by headache. The diagnosis was thrombosis of the posterior cerebral artery with a softening of the medulla. There was a history of syphilis. Dumeneil[41] has written an elaborate article on unilateral paralysis of the soft palate, citing twelve cases in which he observed this symptom.

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[33] London Hospital Reports, vol. i., 1864, p. 361.

[34] Op. cit., p. 368.

[35] London Hospital Reports, vol. iv., 1867, p. 314.

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[36] Op. cit., p. 318.

[37] Des Paralyses bulbaires, Paris, 1875, History 23.

[38] Arch. f. Psychiatrie, vol. xi.

[39] Loc. cit.

[40] Op. cit., p. 305.

Case eight is worthy of note in that an autopsy was performed, the patient dying of cancer of the pylorus, which revealed an area of softening in the right restiform body about the size of a pea. In this case the paralysis was confined to the palate alone. Eisenlohr[42] refers to three cases which came under his observation, showing a paretic condition of the cords and impaired voice. In one case there was thrombosis of the left vertebral artery and degeneration of the left pyramid and of the right to a certain extent, with sclerosis of the ependyma of the floor of the fourth ventricle. The second case showed atheromatous degeneration of the arteries of the base, narrowing of the vertebral, obliteration of the small vessels running from the basalar arteries to the pons, and degeneration of both pyramids, especially of the left. In the third case, a male patient, aged thirty-three, suffered from complete recurrent paralysis of the left side of the larynx, paralysis of the left side of the palate and left side of the pharynx, voice not affected. There was diminution of sensibility of the skin over the left trigeminus distribution the facial, motor portion of the fifth, and hypo-glossal nerves were intact. The post-mortem showed the left recurrent involved in a thickened pleura and degeneration of the bulb and upper part of the cord. Sokaloff[43] cites the case of a woman, thirty-five years of age, suffering from weakness of the right arm and leg, defective hearing, face drawn to the left, difficulty in swallowing, deviation of the tongue and uvula to the right, and paralysis of the left vocal cord. Later in the disease there was dyspnœa. The autopsy showed a glioma involving the left side of the pons, medulla and olivary body. Ollivier d'Angers[44] cites the case of a male, sixty-eight years of age, suffering from bilateral paralysis of the abductors, dysphagia, spasmodic attacks of coughing, paraplegia and weakness of the arms. The autopsy revealed an aneurism of the basilar artery, compressing the olivary body and the nuclei of the glosso-pharyngeal, vagus and hypoglossal nerves. Gerhardt[45] cites a case of left posticus paralysis with carcinoma, compressing the medulla. A similar case is reported by McBride, as cited by Gottstein,[46] in which carcinoma at the base of the skull gave rise to paralytic symptoms involving the hypoglossal and the glosso-pharyngeal nerves of the left side, left abductor paralysis and complete anæsthesia of the left half of the larynx. A somewhat similar case is reported by Nothnagel,[47] in which an abscess originating in the petrous portion of the temporal bone gave rise to paralysis and atrophy of the left side of the palate, anæsthesia of the left half of the larynx, abductor paralysis of the left vocal cord, dysphagia and paresis over the distribution of the left facial, abducens and trigeminus. Krause[48] mentions one case of abductor paralysis necessitating tracheotomy. Death occurred apparently from suffocation. The autopsy showed a clot in medulla at the nucleus of the vagus and spinal accessory nerves.

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[41] Arch. Generales de Medicine, 1875.

[42] Deut. Med. Woch., 1886, p. 363.

[43] Loc. cit. [44] Loc. cit.

Perhaps no chapter in neurology better illustrates the point which we desire to emphasize, namely, that a laryngeal paralysis should always call our attention to the fact of its probable source in the medulla, than the clinical history of locomotor ataxia or tabes dorsalis. Duchenne, Fereol,[49] and Charcot[50] lay special emphasis on laryngeal crises, or cough and dyspnœa, as not infrequently accompanying the early stages of the disease, although not defining the specific condition observed on laryngeal examination. This point was still further and very fully elaborated by Cherschevsky,[51] who still failed to describe definitely the laryngeal conditions. That the special condition should be so long overlooked is perhaps not remarkable when we consider, as Gottstein[52] suggests, that unilateral paralysis of the abductors may give rise to no special symptoms with reference to the voice.

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[45] Op. cit. [46] Op. cit., p. 311.

[47] Wien. Mediz. Blatter, 1884, No. 9.

[48] Neurologische Centralblatt, 1885, p. 543.

[49] Gaz. Hebdommadaire, 1869, No. 7, p. 108.

[50] Progres Medicale, 1879, No. 17, p. 319.

Krause,[53] in reporting three cases of tabes, gives a definite description of the laryngeal conditions as revealed by examination. In one of these cases there was anæsthesia of the lining membrane of the larynx and loss of reflex irritability. In the other two reflex was retained. In all these cases the cords were paralyzed in the median line. Krause explains this condition on the theory of spasm. As before stated, this view I regard as untenable, the condition being one of genuine paralysis of the abductor muscles, as illustrated in the case reported by Ross,[54] in which the characteristic symptoms of tabes were present, and an autopsy revealed degeneration of the medulla in the region of the columns of Goll and in the posterior nuclear zone and in the cerebellar crura. In the case reported by Kehler,[55] there was difficulty in swallowing, the right vocal cord was in the cadaveric position, the left was normal, there were also attacks of coughing, pharyngeal paralysis, and paralysis of the closers of the epiglottis. The autopsy showed ependymitis of the floor of the fourth ventricle, with sclerosis involving the vagus nucleus, especially the right. Oppenheim[56] cites a case of paralysis of the right side of the palate, right vocal cord, the ocular muscles and paræsthesia of the right lower extremities with loss of knee-jerk. Electrical stimulation of the right recurrent nerve in the neck did not cause abduction of the vocal cord, although a similar experiment performed on the left side was successful. This experiment would seem to indicate degeneration in the trunk of the nerve, and yet this would not necessarily contra-indicate disease of the medulla. No autopsy was made in this case. Jean[57] cites a curious case of laryngeal cough with spasm of the pharyngeal muscles, the autopsy showing atrophy of the restiform body and posterior pyramids, atrophy of the filaments of the left pneumogastric and spinal accessory at their origin. Wegner[58] is quoted as reporting two cases of his own and making a compilation of other cases of tabes with laryngeal paralysis, in all of which the laryngoscopic lesion was the same, namely, paralysis of the abductor muscles. In a case reported by Semon,[56] of bilateral paralysis of the abductors, the patient developed well-marked symptoms of tabes two years later. Semon takes the ground that there is no necessary connection between the two, and attributes the paralysis to other causes. In the light of later reports this position is hardly tenable, laryngeal paralysis marking more probably the early development of spinal disease. Thus, in the first of my own cases the tabes did not develop for more than two years after the laryngeal symptoms set in. Saundby[60] reports a case of tabes in which there was paralysis of the abductors and paresis of the adductors with abnormal frequency of the heart's action. On autopsy, there was degeneration of the posterior fibres of the cord extending into the medulla, involving the posterior pyramids and restiform bodies. The recurrent nerve was in a condition of chronic interstitial neuritis.

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[51] Revue de Med., 1881, No. 1.

[52] Op. cit, p. 323.

[53] Berlin. Klin. Woch., 1886, No. 20, p. 651.

[54] Brain, London, 1888.

[55] Zeitsch. f. Heilkunde, 1881, p. 440.

[56] Loc. cit.

[57] Loc. cit.

We find thus in examining the literature of this subject from a purely clinical point, how strong the evidence is that a very large portion of cases of laryngeal paralysis have their origin in the medulla oblongata. Experience teaches us that the physiology of the brain is best studied through an investigation of pathological conditions. Certainly as far as the question of localization of function is concerned, by far the most valuable part of our knowledge of this subject is derived from a study of pathological conditions.

The question suggests itself in this connection then, as to how far we are justified in looking for a motor centre of the larynx in the cortical substance of the brain. In none of the cases above reported was there discovered any evidence of a cortical lesion, and yet we find the various muscular movements of the larynx, as well as sensation, completely abolished by morbid conditions confined entirely to the medullary tissues.

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[58] Annual of the Universal Med. Sciences, vol. i., p. 89.

[59] Clinical Society Trans., vol. xi., p. 141.

[60] Birmingham Med. Review, Dec., 1886.

I think if one studies closely the case reported by Delavan[61] and compare it with many of the cases above alluded to, the opinion certainly is warranted that Delavan's case was a bulbar clot rather than infusion into the brain substance proper. I can hardly justify his conclusion therefore that it establishes the existence of a motor centre of the larynx in the third frontal convolution of the brain. Krause,[62] following the same line of investigation, made a series of experiments on lower animals, in which by irritating the surface of the brain at the base of the præfrontal convolution, he produced phenomena which would seem to lend weight to Delavan's theory, but when he resorted to more vigorous measures in destroying the brain tissue, his results were far from satisfactory. Masini[63] endorses this opinion of Krause without, however, substantiating his view by reporting any experimentation on his own part. I think in estimating the value of Krause's experiments it is necessary to bear in mind the statement already made of the very great difficulty of imitating the hidden processes of disease by the coarse and bungling methods of physiological investigation.

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[61] N. Y. Med. Record, Feb. 14th, 1885, p. 178.

[62] Arch. f. Anatomie und Physiologie. Phys. Abtheil, 1884.

[63] 12th Congress of Italian Physicians and Surgeons.